**PURI TRIAGE FOR UNIT 2 ORGAN SYSTEMS TEST**

12/12/12

* Cardiac output
* Majority of blood volume resides in veins—blood is not sitting anywhere. Volume is diff from flow—flow is function of volume. All of the blood is in circulation, it is not sitting anywhere.
* Flow is the movement of volume
* Be able to figure out which segment has highest velocity, resistance, CSA—combined & individual (anatomical characteristics of the vasculature).
* CO = P/R = ΔP/R—don’t forget the *change* in P (but we usually ignore RAP here because so low).
  + May have to calculate change in P on exam
* CV system maintains constant MAP.
* CO = HRxSV
* ΔP is important (MAP-RAP), not absolute pressure.
* Pa-Pv/R = flow in CV system (CO)
  + Pa= MAP and Pv= RAP
* MAP is single most important regulated variable.
* PRESSURE PROFILES! Not the numbers, but their characteristics. **Highest P: LV**. **LV the Diastolic P close to zero**. Aorta- the diastolic P is not close to zero. RV—lower P than LV but still the lower pressure is zero? Pulmonary artery does not have a zero diastolic P. The diff btwn ventricles and large arteries—the lower pressure in ventricles is zero or maybe 5 but the large arteries does have that, the large arteries have recoil. The large arteries and the ventricles have a higher and a lower pressure but capillaries and arterioles do not. Capillaries and arterioles have continuous flow, their pressure profile doesn't read systolic by diastolic, their pressure profiles read just one pressure.
* Pulse pressure—higher PP is indicative of reduced aortic compliance.
* r4 is most important regulator of resistance, but viscosity is also important. Anemia—↓ R. Polycythemia--↑ R.
* more vascular beds there are in parallel—↓ R
* more vascular beds there are in series--↑ R
* Series vs. parallel.
* **Resistance of any organ is higher than TPR.**
  + **Because TPR the organs are in parallel it is lower than individual organs**
* to calculate MAP: need CO and R. that’s it.
* largest pressure drop is across arterioles because highest resistance
* When we talk of Flow as the CO we need (highest pressure in aorta – lowest pressure in the RA) / combined TPR of all vascular beds
* Flow of blood through an organ = (Arterial P=MAP - Venous Pressure)/Restistance of that organ only
* Ex: Higher than normal MAP (should be thinking about baroreceptors—but wont be a choice on the exam) will ask you a consequence of the baroreceptors. Or think about PN curve
* LOOK AT EXAMPLE QUESTIONS.
* velocity reduced/increased by? Atherosclerosis🡪↑ Velocity b/c ↓ CSA 🡪 ↑ turbulent flow (↑ perceived resistance)
* Condition associated w/ high turbulent flow: **aortic stenosis (most important), PDA**, AV fistula, VSA—turbulent flow, associated with very high velocity flow
* COMPONENTS OF VASCULAR WALL—elasticity (aorta), VSMC (arterioles—contracting SM🡪↓ elasticity)
* contract SMC—even lower elasticity
* Veins are similar in structure to aorta but veins are **collapsed**
* compliance curves! as you ↑ P in veins (even a little b/c of their compliance ↑ V. ↑ V🡪blood will pool.
* SNS changes elasticity of arteries—**elasticity of arteries ↓ with SNS stimulation.**
* Windkessel effect—function of aortic recoil; Aortic recoil has another function 🡪 it limits rise in systolic and limits the drop in diastolic (compliance is ↓ --then systolic is higher than what it should be and diastolic is lower than what it should be🡪 systolic HTN, MAP may be same)
  + 120/80 gives same MAP as 192/47
  + ↓ aortic compliance is seen in: aortic fibrosis, aterial sclerosis, loss of elastic tissue and increased collagen deposition
* aortic compliance is the reason that LV has diastolic of zero whereas aorta does not.
* (PA has same structure as aorta) so PA also has recoil…has compliance. may be pulmonary fibrosis (instead of aortic—same result)
  + large arteries have recoil
* Pressure Wave Velocity is a measures aortic compliance (Don't need to know how to calculate). Higher PWV—lower compliance. Lower PWV—better compliance.
* Endothelial function. **Nitric oxide is a vasodilator**. NO is released by endothelium.

12/13/12

* Factors regulating filtration at level of any capillary
  + forces in favor of filtration—cap hydrostatic pressure
  + forces against filtration—oncotic pressure
  + The question will not ask you to predict filtration and give you pressures. The question will you give pathology and then have you predict the pressures
* how does arterial tone affect filtration
* how does venous tone affect filtration at capillaries (equation is intuitive, think of as pathologies)
* diseases that cause edema and why (chart)—probably a question about capillary filtration gone wrong
  + if there is a question about capillary filtration it will probably start with edema
* tissue o2 supply—factors can change independently, one compensates for the other
  + know how tissue oxygen supply can be changed independently (BF and extraction)
* vascular smooth muscle contraction—what initiates this? and what is the mechanism (basic)—MLCK has to be p’d, which in turn p’s MLC—essential for contraction!
* membrane voltage changes VSMC tone
* chart—**adenosine**—vasodilation—opens K+ channels
  + know adenosines mechanism of action
* no absolutes in tissue blood flow, it is a balance! If you aren’t given a scenario in which the balance is changed🡪 **assume local metabolic regulation is the strongest** influence if nothing else has changed; more true in heart and brain
* **NERNST EQUATION**—can be same as membrane potential, can be different. assumption: membrane is completely permeable to ion; **hyperkalemia & hypokalemia** (K is the regulator of membrane potential)
  + **Don't confuse nerst potential with membrane potential**
* relative permeabilities of K Na Cl become important if become changed—be able to make accurate **prediction of membrane potential based on K** (not necessarily Na and Cl)
  + in the resting cardiac myocyte Na and Cl can be ignored but an exam question may not ignore them—may be asked to predict something about them
* which tissues have fast potential and which have slow AP—You will be given a tissue and the question will be about what happens on EKG.— so you need to remember type of AP and what are ionic channels conductances & currents of that AP
  + SA and AV have slow
* AP PHASES! Which ions are moving and (**more important) which are not moving** in both fast and slow AP
* Cardiac effective refractory period is a function of **inactivation** of Na channels and Ca channels (not closed), K channels do not do this
* determinants of conduction velocity (related to #1-3: ***rate of rise of phase 0, amplitude of phase 0***, threshold of Na channel opening)
  + will not see CV mentions in respect to cardiac diameter of myocytes
  + they will give you an AP and ask you to predict CV (***rate of rise of phase 0, amplitude of phase 0 are the most important determinants of CV)*** 
    - if the amplitude bigger the CV is more
  + CV is a primary constituent of reentry arrhythmias (so they might give you a reentry arrhythmia and ask you about CV)
  + higher amplitude—higher conduction velocituy
* NO ONE will ask about the phenomena of rectification but they will assume you know which phase of the AP have delayed rectifiers and inward rectifiers
  + which phase of AP has delayed/outward rectifiers (mediate repol—phases 1, 2, 3), which phase of AP has inward rectifiers (phase 4, closed during phase 0)—if at rest on EKG, resting isoelectric line (in between QRS and P waves🡪this is resting membrane potential) predicts that the delayed/outward rectifiers are closed and also another isoelectric line during QT segment—plateau) open during plateau isoelectric line
* AV nodal conduction delay🡪heart block (by looking at EKG and determine what is the state of AV node?)
  + AV node is site of conduction delay so if you have a prolonged PR interval🡪 conduction delay is prolonged
  + If you have 2 P waves and 1 QRS= second degree heart block
  + If you have randomness of QRS and P waves= third degree heart block
  + Question will ask you where pathology is…AV node or the tissue which has this pathology have a 0 phase upstroke mediated by which ion channel? Answer is calcium channel
* Refractory period due to inactivation of Na and Ca channels, recovery from refractory period is a function of closed Na channels; Na channels are ONLY open during phase 0!
* Stronger QRS—faster phase 0 was
  + High CV—CV of phase 0 is dependent on opening of Sodium channels 🡪this will be less if the membrane is partially depol b/c that means some of the Na channels are left inactive
    - So the question will starts with MI…with myocardial ischemia, what effect do you expect on the CV?—Partial depol🡪 ↓ slope of phase 0🡪 inactivation of Na channels🡪 CV will be slower 🡪 unidirectional blood🡪 reentry arrhythmias
    - Reentry arrhythmias: need ↓ conduction velocity and ↓ refractory period
      * Question will be how do you reduce effective refractory period? 🡪 SNS activation🡪 ↑ outgoing K current 🡪 faster Repolarization🡪 effective refractory period is shortened
* Autonomic control of HR—phase 4 (HCN channel—funny channels—regulated by cyclic nt= cAMP they are proportional) and slope of phase 0 (L-type Ca channels)
* Don’t need to know subtypes of inward or outward rectifiers
* Which direction does Na/K ATPase work? (“electrogenic pump” **charge follows Na**—same true for NCX!)
  + They will give you the direction of working of the NCX and ask you the effect on membrane potential (if + is coming in—depol; if + is going out—repol)
* Block Na/K ATPase (as in MI)—NCX will pump out Na and charge will follow Na
* Early afterdepol—prolongation of QT (phase 2 prolonged) on EKG
  + Which ionic channel isn’t working properly? Phase 2 is plateau phase—K channel controls this
    - Prolonged QT🡪 ↓ in K activity and vice versa
* No vector calculations on EKG—questions on EKG will be about action potential—know what parts of AP are seen on EKG (red graph)
* How is EKG affected by size of heart, obesity, air in lungs, etc.
* If they give you a PR interval they will give you a normal (PR interval is just AV conduction)
* No one will ask ST elevation
* Path of repolarization—part of myocardium that depols first repols last (has longest AP) 🡪 endocardium
* **HEART BLOCK** slide!
* Regulation of myocardial contraction—TnC—binds Ca; TnI—inhibitory; movement of TnI along with Tropomyosin unlocks the mechanism
* No A band H band stuff
* No cycle of contraction
* Majority of Ca from SR, majority of Ca back to SR through SERCA (know all channels on that slide, question related to a drug blocking a channel)—contractility is function of Ca
  + They will give you a drug that affects myocardial contractility (function of calcium)🡪 ↑ contractility means ↑ Ca and vice versa
* Frank-Starling is the same as Length-Tension curves
* No sliding filament theory
* No LaPlace law—its very confusing and not many questions
* **CARDIAC CYCLE**—pressures are important! when are chambers filling, when are valves closed
  + Mitral valve opens at the end of isovolumetric relax or passive ventricular filling
* **WIGGER DIAGRAM**—important: pressure difference (P in ventricles is not the same as P in aorta—especially diastolic🡪 in ventricles diastolic is 0 in the aorta the diastolic P is 80
  + This concept will appear in context of a valvular heart dz---will ask what is the LVP in aortic stenonis, regurg, mitral stenosis, etc with regards to aortic pressure)
* **Indices of cardiac preload**! **PCWP and LAP are most frequent**
  + PVP= preload
  + LVEDP= preload
  + PCWP= preload
* Preload is indep of contractility, contractility is indep of preload; You can change one without changing the other or can change both but they are indep
* **P-V LOOPS!!! guaranteed question on Puri’s exam**
  + ESPVR represents contractility (slope of line)
    - steeper slope—more contractility
      * if that all have the same slope then same contractility even if they look diff
    - exam will ask about ejection fraction is a measure of contractility
    - reduced slope (reduced ESPVR) in heart failure
    - change—**systolic dysfunction**
  + EDPVR represents compliance (slope of line)
    - changed in **ventricular fibrosis🡪 ↓ compliance**
    - change—**diastolic dysfunction**
  + ONLY LOOK AT SLOPE
* More Ca leads to more contractility.
* **Afterload is the peak ventricular pressure**—max pressure ventricle generates (not the aortic pressure but they may be the same); might see increased afterload even if AV opens at same time (this would mean you have the same diastolic P)—.
* Causes of higher afterload → reduced SV, reduced ejection
  + aortic stenosis, HTN, and ↓ aortic compliance all 🡪 ↑ afterload
* 3 regulators of SV
* Recognize systolic and diastolic dysfunction, don’t need to know diseases
  + Diastolic dysfunction (smaller loop within the loop)
  + If ventricular compliance is ↓ the EDV is ↓ b/c pressure in ventricle is higher and they can’t fill enough
* **Double product**: ↑ HR X ↑ Arterial P ⇒ higher work of heart🡪 more energy the heart needs
  + Coronary perfusion is product of HR and Arterial P
  + question related to coronary blood supply—increase cardiac blood flow to ↑ O2 to heart b/c it is already at maximum O2 extraction
* Aortic stenosis, aortic regurg, mitral stenosis
  + systolic murmurs (rules out mitral stenosis) vs. diastolic murmur (rules out aortic regurg)
* **REGULATION OF MAP**—most questions!
  + short term—high P baroRs
    - DON'T FORGET THEIR ADAPTABILITY
      * chronic elevation of MAP—high P baroRs aren’t influential anymore.
* Relay center = medulla (don’t need to know much more about that slide)
* Don't need to know mechanism of contraction but do remember PSNS tone is predominant on heart, SNS tone is predominant on vasculature
* Acute nervous regulation of MAP (flow chart slide)—don’t forget venoconstriction (↑ venous return)
* Venous pooling flow chart
* EKG in response to neck pressure (not on Puri but maybe boards)
  + Predict what initiated that response
  + Acute responses to HPBR are working
* Chronic regulation of MAP—Renin regulation (signals affecting renal secretion, question will not be on these signals but on proximal to these signals (like SNS)—like HPBR.
* Don’t worry about angiotensin II effects; just now angiotensin II causes vasoconstriction and ↑ Na/H2O retention
  + Question will be on Renin on angiotensin II (they are like the same thing)
* What does P-N curve regulate? ECF volume by changing Na excretion in response to chronic change in MAP; don’t worry about curve shifts right now.
* Arms of MAP regulation (important slide)—RAAS affects both arms🡪ST (acute) and LT (chronic)
  + Acute—vasoconstriction
  + Chronic—sodium retention
* ↑ TPR is a consequence of essential HTN not a cause
  + if you have ↑ than normal MAP the vasculature in the kidney, brain, heart, and GI (all doing autoregulation)—they are all effectively ↑ TPR and this feeds back on the MAP
  + not on exam
* CO—things that affect CO
* HR and preload changed by sympathetic nervous system (venoconstriction)
* ANP—stretch on the atria activates low pressure baroRs more importantly ANP—activated regardless of MAP, activated by volume (stretch)
* Cardiac and vascular function curve (guaranteed on exam)—for CO or VR to change, shape of curve has to change! SNS has capacity to change both by ↑ contractility for CO curve, ↑ VR for venous function curve
* Vasodilation is in arterioles!
  + Changes the slope of the VR curve
  + Venoconstriction is in the veins—it doesn't change the slope but moves the curve to the R
  + ON SLIDE—change “role of venodilation” to “venoconstriction”
    - venousconstriction causes parallel shift in curve
* Exercise—TPR is lower than normal (vasodilation)🡪 SNS is active and leads to constriction on venous side (↑ venous return), dilation on atriolar side—not because of SNS but because of falling resistance in the muscles (local metabolic factors)
  + SNS tries to constrict the arterioles but fails b/c the arterioles in muscles are dilating themselves
  + Changes in cardiac curve
    - Usually where question will start
    - Heart failure—shift down and right
* Most important circulation in “special circulation” is coronary circulation
  + Tachycardia ↓ cardiac perfusion but cutting into diastole
  + Higher MAP ↓ cardiac circulation by preventing flow during systole
* Exercise slide!